Short Communication

Successful Treatment of Diabetes Insipidus with Oral Desmopressin

Syed Nayyer Mahmood, Muhammad Ali Afzal, Saman Zafar, Faisal Muhammad

From Department of Medicine, Shifa International Hospital, Islamabad.

Correspondance: Dr Saman Zafar, Shifa International Hospital, Islamabad.
E-mail: samanjzafar@hotmail.com

Received: February 3, 2005 Accepted: April 2, 2005

ABSTRACT

Acute-onset polyuria, with urine volumes exceeding 3 litres per day, is a relatively uncommon clinical condition. If managed on appropriate guidelines, an excellent recovery is expected. (Rawal Med J 2005;30:46-47).

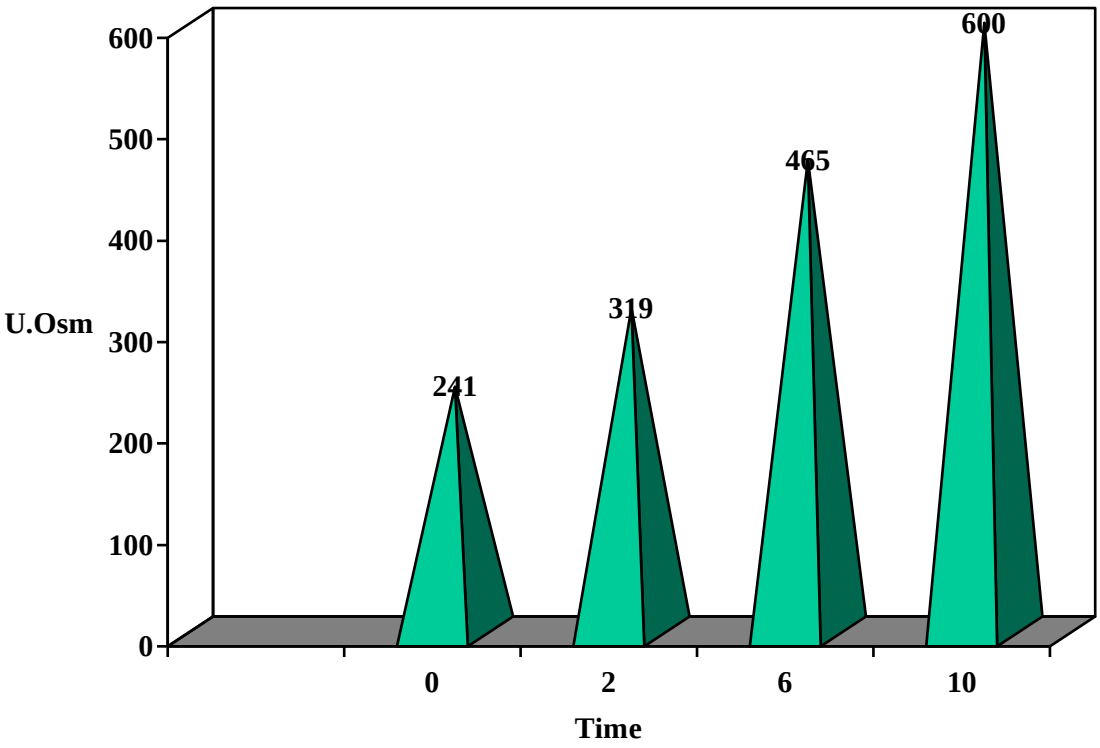
Key Words: Serum/ Urine osmolarity, diabetes Insipidus.

CASE SCENRIO

A 37 year old male presented to our OPD with increased urinary frequency and thirst for last six months. He was urinating 13-14 times a day, 4-6 times at night, and consuming 30-40 glasses of water daily. The general and systemic examinations were unremarkable. His routine laboratory and radiological investigations were normal, except for a serum sodium concentration of 149mEq/L. Routine urine report showed a specific gravity of 1.003. Urine osmolality was 91mosm/l. Calculated serum osmolality was 288mOsm/L and spot urinary sodium was 70mEq/L.

The patient was presumed to have diabetes insipidus (DI) and was placed on the water deprivation test for 6 hours. At the end of the test, his serum sodium was 150mEq/L, serum osmolality 300mOsm/L and urine osmolality 80mEq/L. For confirmation of DI, he was given two tablets of Minirin (desmopressin 0.1mg), after which the urine osmolality was assessed at time intervals of 0,2,6 and 10 hrs. The progressive increase in the urine osmolality is shown in Fig.1.

Fig-1. Changes in U.Osmolality in responce to Desmopressin



The patient was placed on 0.2 mg of Desmopressin orally daily. His serum sodium after 72 hours was 143mEq/L, serum osmolarity was 280mOsm/L and urine osmolality was 235m Osm/L. On follow up 2 weeks later, he reported resolution of his initial symptoms. Serum sodium was 146mEq/L, serum osmolality was 288mEq/L and urine osmolality was 290mEq/L. MRI of brain was reported normal.

DISCUSSION

Polyuria can be arbitrarily defined as urine output exceeding 3 L/day. Three major causes of polyuria in the outpatient setting are primary polydipsia, central diabetes insipidus and nephrogenic diabetes insipidus. In patients presenting with acute onset of polyuria, differentiating between these 3 conditions is usually possible by analyzing the history and checking the serum sodium concentration (table-1). A low plasma sodium concentration at presentation (less than 137 meq/L) is usually indicative of primary polydipsia, whereas a high-normal plasma sodium concentration (greater than 142 meq/L) points toward DI[1]. Patients with DI usually compensate for increase urinary water losses by maintaining a higher water intake, and as long as their thirst mechanism is adequately stimulated, the serum sodium concentration may not rise substantially.[2]

Objective proof of the diagnosis rests on correct performance and interpretation of the water restriction test. Patients should stop drinking two to three hours before starting the test. Overnight fluid restriction can potentially cause severe volume depletion and hypernatremia in patients with marked polyuria, and should be avoided.

Table-1. Diagnosing central diabetes insipidus with the help of serum Sodium Concentration and the Water Restriction Test. (ref. [3]).

Parameter	Primary Polydipsia	Central Diabetes Insipidus	Nephrogenic Diabetes Insipidus
Initial Serum Sodium Concentration and plasma osmolarity	Low	High-normal/high	High normal
Effect of water restriction on plasma osmolarity and sodium	Slowly rises to normal	Rises	Rises
Effect of water restriction on urine volume	Falls to normal	Remains high	Remains high
Effect of water restriction on urine osmolarity	Rises to >600mOsm/kg	Fails to rise >600mOsm/kg	Fails to rise >600mOsm/kg
Effect of dDAVP on urine osmolarity	No effect/ falls	Rises to >600mOsm//lg	Fails to rise to >600mOsm/kg.

Desmopressin, a two-amino acid substitute of ADH that has potent antidiuretic but no vasopressor activity,[4] is the appropriate pharmacological treatment of central DI. The intranasal daily maintenance dose is about 5 to 20 µg once or twice a day. The oral form has about one-tenth to one-twentieth the potency of the nasal form because only about five percent is

absorbed from the gut. Thus, a 0.1 mg tablet is the equivalent of 2.5 to 5 µg of the nasal spray.[5] Both forms of the drug are equally expensive.

There are few long-term data on the use of the tablet form of desmopressin. In one study, eight children with central DI were treated and followed for up to 3.5 years.[6] There was no attenuation of the antidiuretic effect and no side effects or antibody formation was noted. In another report, ten adults had satisfactory maintenance of the antidiuretic effect over one year with doses of 0.3 to 0.6 mg/day given in two to three doses per day; doses larger than 0.2 mg had no greater effect, e.g., 0.4 versus 0.2 mg, but probably lasted longer.[5]

In our patient, the results of the water deprivation test were simple to interpret, and the standard treatment of central DI with oral desmopressin 0.2mg/day was adequate to relieve the patient of symptoms, as well as return his biochemistry to normal.

In conclusion, the diagnosis and management of polyuria, if done with precision, can be a rewarding experience for both the patient and the clinician.

REFERENCES

1. Rose, BD, Post, TW, Clinical Physiology of Acid-Base and Electrolyte Disorders, 5th ed, McGraw-Hill, New York, 2001, pp. 748-757,767-772.

2. McIver, B, Connacher, A, Whittle, I, et al. Adipsic hypothalamic diabetes insipidus after clipping of anterior communicating artery aneurysm. BMJ 1991;303:1465

3. Zerbe, RL, Robertson, GL. A comparison of plasma vasopressin measurements with a standard indirect test in the differential diagnosis of polyuria. N Engl J Med 1981;305:1539.

4. Richardson, DW, Robinson, AG. Desmopressin. Ann Intern Med 1985; 103:228.

5. Lam, KSL, Wat, MS, Choi, KL, et al. Pharmacokinetics, pharmacodynamics, long-term efficacy and safety of oral 1-deamino-8-D-arginine vasopressin in adult patients with central diabetes insipidus. Br J Clin Pharmacol 1996;42:379.

6. Fjellestad-Paulsen, A, Laborde, K, Kindermans, C, et al. Water-balance hormones during long-term follow-up of oral DDAVP treatment in diabetes insipidus. Acta Paediatr 1993;82:752